Original Article

# Results of diplopia and strabismus in patients with severe thyroid ophthalmopathy after orbital decompression

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**Purpose:** It has been frequently stated that the orbital decompression, in patients with thyroid ophthalmopathy, does not usually improve extraocular muscles function and that after the operation there is often a deterioration of these functions. The purpose of this article is evaluation of extraocular muscles function after applying personal method of 3 wall orbital decompression. **Materials And Methods:** Retrospective review of case records of 119 patients with severe thyroid ophthalmopathy seen and treated by the author between December 1986 and December 2010. All patents underwent 3 wall orbital decompression combined with removal of the periorbital, intraorbital and retrobulbar fat. Correction of coexistent eyelid retraction and deformities were also performed. **Results:** Comparison of preoperative and postoperative results was conducted in 65 patients three months after 3 wall decompression. All patients showed a significant reduction of exophthalmos [5-11 mm, 7.2 mm on average], reduction of intraocular pressure, marked improvement in ocular muscle function as well as considerable reduction in or disappearance of subjective symptoms. There were no cases of subsequent impairment of ocular motility. Strabismus surgery was performed in 6 patients with residual diplopia. There was an improvement in vision in 68% patients who had impaired vision before the operation. Less evident relapse of exophthalmos was recorded in 3 cases only and only one patient required unilateral reoperation. **Conclusion:** It can be concluded that this method of orbital decompression is logical, based on an understanding of the pathology, has less complication rates, is relatively easy to perform, gives very good functional and aesthetic long term results and allows rapid recovery.

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Thyroid ophthalmopathy [TO] affects orbital contents and eyelids. Aethiopathogenesis of this disease has not still definitively explained. Besides a number of hypotheses and theories of the prevailing option is that it is actually an autoimmune multifactoral process.[1-4] Numerous articles on the autoimmune pathogenesis and pathophysiology are in many ways speculative and controversial. Controversial are the facts about what is primary in the autoimmune process and what stands responsible for common antigens in the orbit and in the thyroid gland, then about the function of the circulating antibodies against the extraocular muscles and the fibroblast component, etc. Since the conservative therapy of severe TO do not often give satisfactory results, the surgical treatment is used more and more often nowadays. Surgical procedures include operations on the extraocular muscles, operation on the eyelids and different methods of orbital decompression, i.e., removal of different parts and size of orbital walls and orbital fat. Because it has been frequently stated that orbital decompression does not usually improve extraocular muscle function and that after the operation there is often a deterioration of this function,[5-11] the aim of this work is to present postoperative results of extraocular muscles function after applying personal method of 3-wall orbital decompression combined with removal of orbital fat and correction of eyelid retraction and deformities.

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## Materials and Methods

From December 1986 to December 2010, 119 patients with severe TO [exophthalmos 24-33 mm] were treated, by the author, surgically using personal method of orbital decompression along with correction of the eyelid retraction and deformities. There were 91 women of the ages 17 to 73 years and 28 men of ages 22 to 53. Decompression of the orbits was performed after complete endocrine and ophthalmic assessment and treatment. The procedure for the correction begins within the upper eyelid [Fig. 1]. As for a blepharoplasty, the excision of the excessive eyelid skin and swollen subcutaneous tissue is performed and the eyelid fat and the fat of upper part of orbit are removed. In order to obtain the correction of upper eyelid retraction, the central part of the aponeurosis of the levator muscle and Muller's muscle are excised. Following this, an blepharoplasty, subciliary incision is made in the lower eyelid, 2-3 mm below the eyelid margin, and through this, the floor and lateral wall of the orbit are explored and the periorbital and intraorbital, peribulbar fat is removed, as much as possible. Using a small chisel and hemostat, the posterior, retrobulbar part of the orbital floor and the lateral orbital wall, except lateral orbital margin, is removed [Fig. 1]. The bony bridge between the floor and lateral orbital wall defect is removed to produce a large continuous orbitectomy. The size of the defect is related to the severity of the ophthalmopathy and exophthalmos. An attempt should be made to remove the bone from the orbital floor as close to the orbital apex as possible. The anterior, bulbar part of the orbital floor must remain intact. The infraorbiral nerve is protected during the floor osteotomy. This is achieved by removal of bone on either side of nerve and then freeing the nerve. If the bone bridge in the infraorbital nerve region is left, the effect of decompression is significantly diminished. If possible, the sinus

mucosa should not be opened. [Small perforations of the sinus mucosa are frequently unavoidable, but are of no importance]. Wide incisions or excisions of perorbital periosteum should be made in several sites. Following this, through an incision made over the medial margin of the orbit, the medial orbital wall is explored and its ethmoidal part is removed [Fig. 1]. This medial wall bone defect should be in continuity with orbital floor defect, i.e., retrobulbar part of orbital strut should be removed. During the medial wall ostectomy, the bone at the region of the upper border of ethmoid bone must be intact. The bone defect begins about 3 mm below the ethmoidal foramens. This is done in order to prevent injury and bleeding of the ethmoidal arteries and to prevent the breaking into scull cavity and damage dura. Using this same approach, the retobulbar space is explored, and again, as much as possible, retrobulbar fat is removed with a hemostat. The periorbital periosteum in this region should be excised or incised at several sites. In the patients with distended, hypotonic and ptotic lower eyelids it is necessary to perform the lateral canthopexy in order to correct these deformities. In some patients it is necessary to perform shortening of the lower eyelid by excision of central part of eyelid rim, about 3 mm wide. After establishment of satisfactory hemostasis, a thin vacuum drain is inserted into retrobulbar space, and the incisions on eyelids are closed. At the beginning of the operation, temporary blepharorrhaphy should be performed using two single sutures. These sutures are removed 5 to7 days after the operation. Postoperatively the eyesight is controlled through central part of the eyelids, between the two sutures. In all cases, except the 3 cases of unilateral disease, the operation was performed on both orbits in a single operation. After the operation, in different time intervals, in all patients the subjective symptoms were observed, as well as the changes of eyelids, conjunctiva and cornea recorded. Degree of exophthalmos by Hertel, the function of extraocular muscles, i.e., diplopia by Hess-Lancaster test, intraocular pressure, the change in the disc, i.e., the changes in vision and field of vision, as well as the thickening and elongation of extraocular muscles, by CT or MRI scan or echography, done.

## Results

Using described method of orbital decompression, all symptoms and manifestations of TO disappeared or were significantly decreased. Although the results are evident immediately after operation, the definitive results were obtained in 2 to3 months thereafter. The complete comparisons of preoperative and postoperative results were conducted in 65 patients three months after operation. Postoperatively, in all patients subjective symptoms were considerably reduced or have disappeared. The swelling and deformities of the eyelids disappeared or were significantly reduced. Due to unsatisfactory correction of the upper eyelid retraction it was necessary to perform lengthening of the upper eyelid in four patients and correction of lower eyelid deformity in three patients Conjunctival hyperemia and chemosis, as well exposure keratitis got resolved in all patients. All patients showed a significant reduction of exophthalmos [5-11 mm, 7.2 mm on average] with marked improvement or normalization of ocular muscle function [Figs. 2-7, Tables 1 and 2]. There were no cases of subsequent impairment of ocular motality, diplopia and strabismus. Due to unsatisfactory correction of double vision and because of residual diplopia, strabismus surgery was performed in six patients. Out of 129 TO orbits in which decompression was performed, there was impairment of vision in 67 eyes due to optic neuropathy or/and due to corneal change. After the operation, the majority of these patients [68%] showed improvement of vision and improvement or elimination of the field defect [Table 3]. In all patients with partial atrophy of disc and irreversible changes in the cornea, there was no improvement of vision. In only one patient the subsequent unilateral impairment of vision occurred. The intraocular pressure measured by mmHg before the operation was 12 to30, mostly 23 to27, the average being 23. After the operation the pressure was 12 to23, mostly 12 to19, average being 15. Until now, less evident relapse was recorded in only 3 cases out of total patients group and in only one patient was unilateral reoperation required [removal of fibrous tissue from bony defects of the orbital walls, which were done in the previous surgery, as well as eventual widening of these bony defects]. Echography, CT and MRI scans showed great reduction of extraocular muscle thickening and shortening of the retrobulbar cone in all operated patients. Finally, significant aesthetic improvement has been achieved in all patients [Figs. 2-7].

Having followed these patients for years, the already

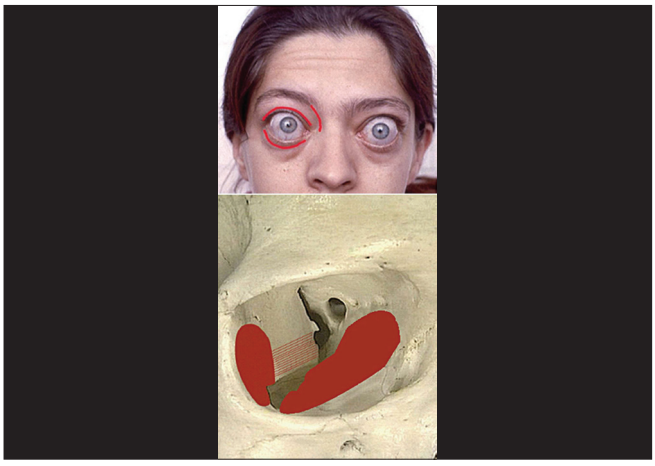

**Figure 1:** The approaches to the orbital walls are marked on the eyelids. The surgical bony defect is marked on the orbital walls

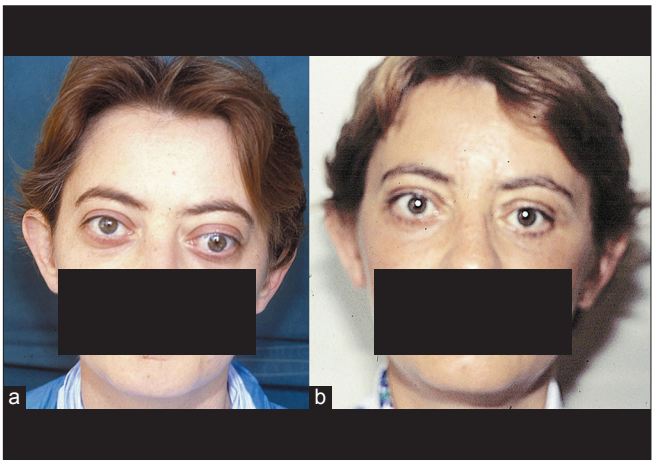


**Figure 2:** Patient with exophthalmos of 28 mm, strabismus, diplopia and eyelid deformities, before (a) and 3 months after the operation (b), without diplopia

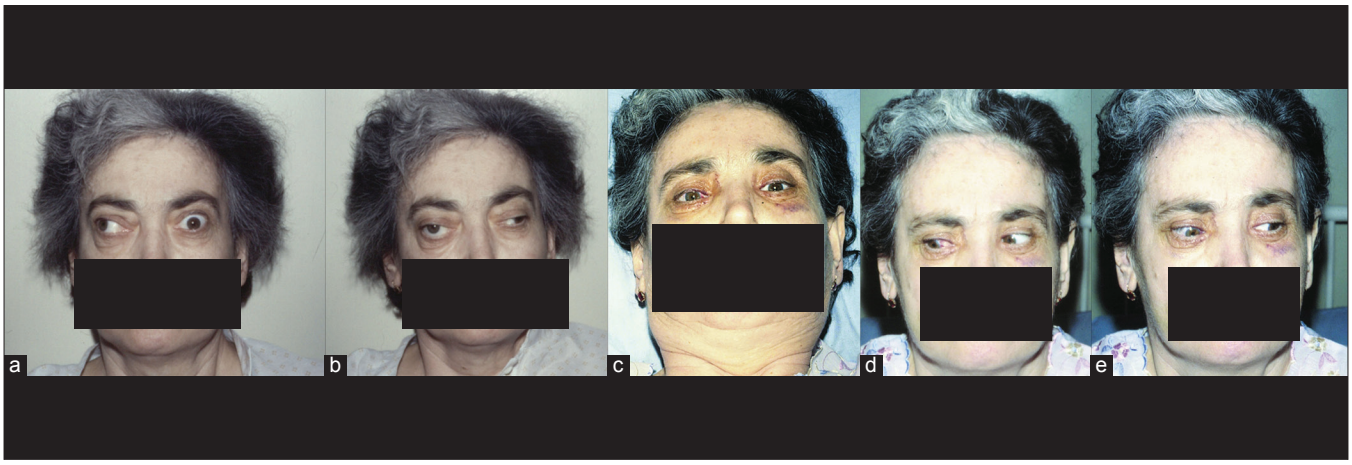


**Figure 3:** Patient with asymmetric exophthalmos of 28 mm and severe strabismus, before (a,b) and 11 days after operation (c, d, e), with residual diplopia on upward gaze

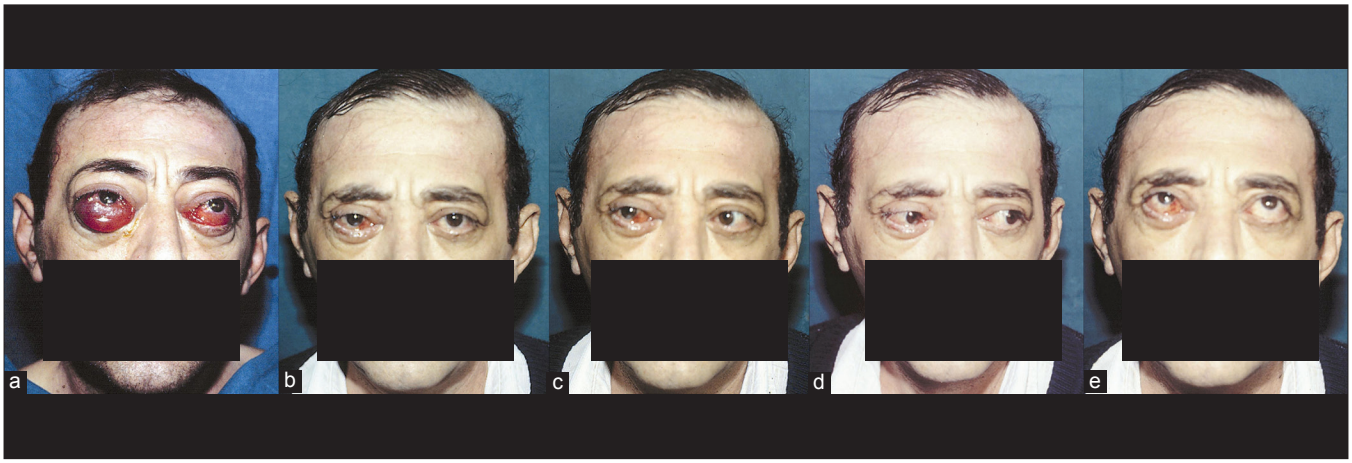


**Figure 4:** Patient with malignant exophthalmos of 33 mm with strabismus before (a) and 3 weeks after the operation (b, c, d, e)

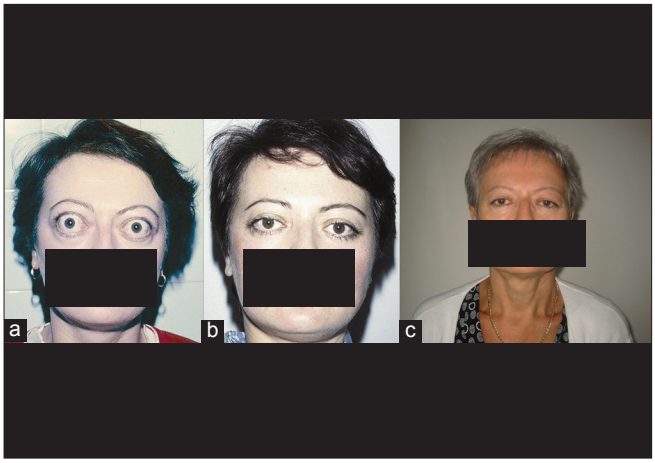


**Figure 5:** Patient with exophthalmos of 30 mm and eyelid retraction, before (a), after 2 years (b) and after 23 years (c), without diplopia

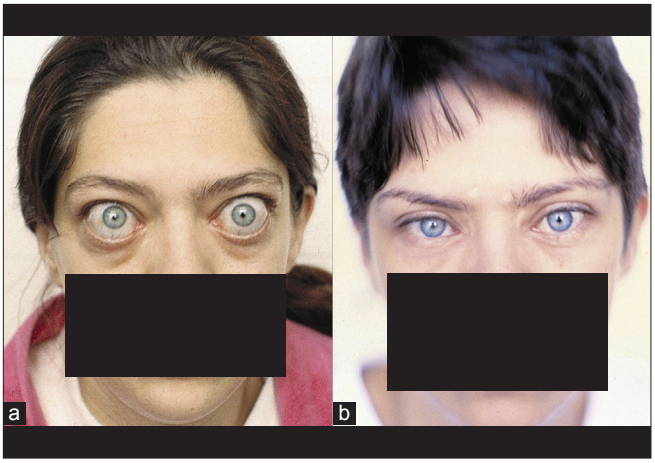


**Figure 6:** Patient after thyroidectomy with exophthalmos of 32 mm and with eyelid retraction and deformities [changes on the eyes are worsened after thyroidectomy] before (a) and 3 months after operation (b), without diplopia

mentioned results, with adequate endocrinolocgical therapy, are stabile.

Complications: The majority of patients had transient hypoesthesia or anesthesia of the infraorbital region. In one patient there was unilateral loss of vision a week after the operation. The ophthalmological conclusion was that the reason for the loss of vision was the embolism of the central artery of

**Table 1: Changes in exophthalmos 3 months after orbital decompression [*n* = 129]**

| Reduction in mm. | Number of eyes |
|---|---|
| 5 | 11 |
| 6 | 24 |
| 7 | 48 |
| 8 | 29 |
| 9 | 11 |
| 10 | 4 |
| 11 | 2 |

**Table 2: Diplopia before and 3 months after orbital decompression [*n* = 65]**

| | Without diplopia | Diplopia on upward or/and lateral gaze | Diplopia on straight gaze |
|---|---|---|---|
| Before surgery | 17 | 33 | 15 |
| After surgery | 40 | 19 | 6 |

**Table 3: Vision changes 3 months after orbital decompression [n = 67]**

| | |
|---|---|
| Improvement | 46 |
| Unchanged | 21 |
| Impairment | 0/1 |

retina. One patient, with chronic rhinitis, had meningitis with good therapeutic results. One patient had the nasal CSF with spontaneous disappearance during one week.

## Discussion

There are still a number of controversies and speculations regarding the ethiopathogenesis and the therapy of TO. It should be noted that there is no excuse for further applications of conservative treatment which does not give satisfactory results. The irreversible changes of optic nerve, cornea and extraocular muscles, i.e., disc atrophy, ulceration of cornea and extraocular muscles fibrosis, should be avoided. If disc atrophy has developed, no improvement in vision can be achieved by orbital decompression. If extraocular muscles fibrosis has occurred, neither exophthalmos nor diplopia and strabismus can be corrected satisfactorily by orbital decompression. Significant symmetrical decompression, particularly in the narrow orbital apex, in the area where there is highest pressure and main problem, is achieved by the surgical treatment described. The elimination of this pressure is necessary for normalization of the function of the orbital structures, especially of the function of the optic nerve and extraocular muscles. In addition, if the eyelid swelling, retraction and deformities are corrected, all signs and manifestations of TO will disappear or will be significantly decreased. If it is performed with precision, this procedure can be performed without high risk.

The main effect of decompression is achieved by widening of the orbit, i.e., by the removal part of orbital walls, especially in its narrow apical region. The removal of orbital fat, without the removal of part of the orbital walls,[12] can not produce satisfactory decompression in cases with marked TO. In certain patients with more evident exophthalmos, the orbital fat is atrophic and degenerated; therefore, the removal of this small amount of the fat does not give a significant decompression and results.[13-16] In some patients, with asymmetric exophthalmos, there is a smaller amount of fat on the side with more evident exophthalmos than on the other side. So, the main cause of exophthalmos are swollen, elongated extraocular muscles.[13-16] If the volume of orbital fat would be dominant for eye position, numerous of patients with severe TO would

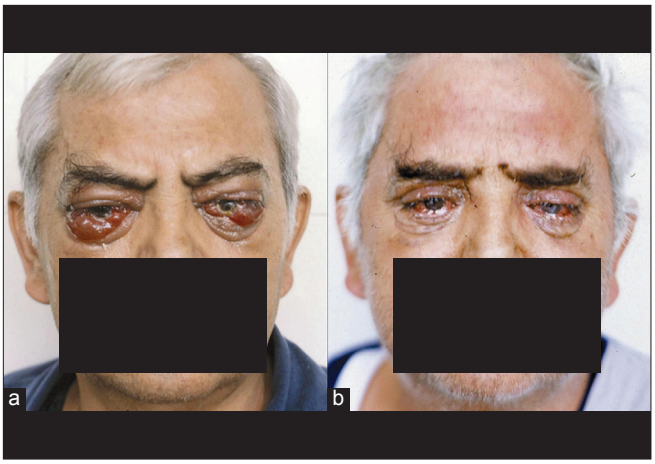


**Figure 7:** Malignant TO with bilateral blindness (a) and 7 days after operation (b). The vision is improved significantly because of recovery of the reversible change of optic nerve and cornea

have enophthalmos due to atrophy of orbital fat. The statement, that is often emphasized, that the removal of 1cc of orbital fat means the reduction of exophthalmos by 1mm, is not correct. Regardless of that, it is necessary to remove orbital fat, i.e., sick tissue, as much as possible. If TO is autoimmune disease, it is possible that main or important autoimmune process occurs in orbital fat.

It has been frequently stated that orbital decompression does not usually improve extraocular muscles function and that after operation, there is often a deterioration of extraocular muscle function. Waller *et al*.[5] emphasize that decompression does not, as a rule, improve muscle function and that muscle dysfunction and diplopia were more apparent after the operation in majority of cases, and that nearly 70% of their total group of patients required one or more strabismus operations after orbital decompression. Shorr *et al*.[6] claim that they had 34% incidence of worsened muscle balance after orbital decompression. Similar statement have been made De Santo.[7] Garrity *et al*.[8] emphasize that after transantral orbital decompression in 428 patients, "postoperatively new diplopia developed in 74 [64%] of 116 patients who had no diplopia before orbital decompression, although 300 patients ultimately had strabismus surgery". Adenis *et al*.[9] report that all patients with moderate or severe diplopia remained with diplopia after fat removal orbital decompression and that in 32% operated patients developed a new diplopia. Russo *et al*.,[10] state that, after 3-wall orbital decompression, in 18% of patients with diplopia there was no modification of diplopia and that in 23,5% of patients a more severe imbalance occurred. Kasperbauer and Hinkley[11] report that only 25% of their patients "did not require eye muscle surgery" after endoscopic

decompression of the orbit. What is the cause of non-improvement and deterioration of extraocular muscle function after orbital decompression? The main cause, beside fibrosis of muscles, is the inadequate and insufficient orbital decompression. TO is complex pathological condition with serious pathological changes of all tissues of the orbital contents and the eyelids. Decompression of the orbit has numerous important details. Even, one inadequate performed detail can be a cause for poor postoperative results, especially regarding diplopia and strabismus. The majority of those who emphasize the poor function of extraocular muscle after decompression of the orbit performed this operation via transantral or endoscopic approach. Applying transantral approach, due to poor visibility in the course of the operation, it is possible to injure the extraocular muscles and can worsen their function. If anterior, bulbar part of orbital floor or its large part is removed, by any approach, the globe loses its support and drops toward the sinus causing marked diplopia and strabismus. Moreover, if the operation is performed in such a way, the pressure in the orbital apex remains, i.e., a sufficient decompression effect, which would enable improvement in optic nerve and extraocular muscle function, is not achieved. In general, if the pressure in the orbital apex is not eliminated, a considerable improvement in extraocular muscles function and optic nerve function cannot be expected. Also, removal of orbital fat contributes to the decrease in pressure in the orbit and the improvement in optic nerve function and extraocular muscles function. In the majority of methods of orbital decompression applied till now, the orbital floor is frequently removed or orbital floor in conjugation with medial and/or lateral orbital wall. Leone *et al*.[17] recommended medial and lateral wall decompression with removal of the lateral orbital rim. They claim that the removal of the orbital floor causes postoperative complications "involving extraocular muscle imbalance, infraorbital anesthesia, hypo-ophthalmia and recurrent sinusitis". Decompression of the lateral wall of the orbit produces a relatively small effect. Removal of the lateral orbital rim does not lead decompression of extraocular muscles and optic nerve in apical region of the orbit, but there is always a visible deformity. If only posterior, retrobulbar part of orbital floor is removed, it is practically impossible to produce hypo-ophthalmia. If orbital decompression is performed with precision, residual diplopia is the consequence of the initial disease process and not of the operation. The same will occur by decompression of only the medial and lateral walls if the preoperative exophthalmos was marked with diplopia. In such cases, there will be more frequent residual diplopia because of insufficient decompression. If the surgery is performed carefully it is quite possible to avoid injury of the infraorbital nerve. After decompression of the orbital floor, if the sinus mucosa is injured, the sinus will be filled with blood. Blood clots in the sinus will be eliminated spontaneously through the natural ostium which happens after facial bone injuries and orthognathic surgery. None of the patients from the group presented has had sinusitis so far. Thus, there is no reason to avoid decompression of orbital floor, i.e., removal of retrobulbar, posterior part. Without this procedure it is impossible to achieve adequate decompression of the orbit, especially of orbital apex, and it is mostly impossible, in marked TO, to get good functional and aesthetic results. If only the lateral part of the orbital floor is removed, using Bartelena[18] and Krastinova[19] methods, it is equally impossible to achieve good orbital decompression since the medial part of orbital floor [medially from infraorbital nerve] is considerably larger than the lateral part. In some cases, in order to perform orbital enlargement and better correction of exophthalmos, Krastinova *et al*.[19] inserted the calvarial bone transplants on the lateral and superior orbital rim and the nasal bone. The mentioned enlargement of orbit makes AP diameter of the orbit longer. Longer diameter of the orbit makes stronger pressure in apical region of the orbit which is certainly unfavorable for further course of disease and diplopia. Apart from decompression in the medial orbital wall, Pearl *et al*.[20] performed osteotomy of part of the lateral and lower orbital wall in the anterior part and the whole bony segment is rotated laterally. It is true that by applying this procedure the orbit is widening in anterior part and its volume is increased, but this procedure gives a poor decompression effect on the orbital apex where the main problem lies. Similarly, orbital expansion by Tessier[21] and Wolfe,[22] along with the visible face deformity, does not give satisfactory decompression in the orbital apex. Though lateral decompression gives the least effect of decompression, in all the cases of decompression through the floor and medial wall, decompression through lateral wall should also be performed. Lateral decompression, in addition, prevents superfluous medial globe displacement which can also be a cause of worsening of diplopia and strabismus.The openings made in all orbital walls should be continuous, without bone bridge between the walls, as well as in the region of infraorbital nerve. In this way an important symmetrical decompression is achieved which enables the recovery of the all orbital structures and balanced decompression, i.e., the recovery of the extraocular muscles. Unbalanced recovery of the extraocular muscle can worsen diplopia and strabismus. It is difficult to believe that four- wall decompression[23] has a significantly greater decompression effect in comparison with three-wall decompression, considering that dura, i.e., the brain, lies against the opening in the orbital roof and could be the cause for pulsate eye or pulsate exophthalmos. Also, it is difficult to believe that this method can produce the globe retrodisplacement up to 16 mm,[23,24] i.e., up to 17 mm.[6] Globe retrodisplacement depends not only on bony openings, i.e., increased orbital volume, also, on the amount of periostal openings and condition of orbital tissues, especially of the condition of extraocular muscles. In order to obtain the functional aesthetic effect of decompression, even in severe cases of TO, it is enough to achieve globe retrodisplacement ranging from 7 to 9 mm which can be done using the method described. Globe retrodisplacement of 16 and 17 mm almost always leads to marked enophthalmos and malposition of the eyes with strabismus and diplopia. Antral-ethmoidal decompression[25,26] and all other previously used methods[27-30] are archaic methods because of poor visibility during operation and poor results. Edoscopic transnasal or transantral orbital decompression is less traumatic procedure than classic surgical methods of orbital decompression. But, with this technique it is impossible to perform wide three-wall decompression and the removal of significant amount of sick orbital fat, i. e., it is mainly impossible to achieve good results in cases of the marked TO. After the endoscopic decompression of the orbit, the patients have often worsening diplopia and strabismus. The transconjuctival decompression has the similar disadvantages as the endoscopic decompression.

Strabismus operations should not be performed before orbital decompression. If orbital decompression is not performed before strabismus operation, the later will only lead to temporary improvement in extraocular muscles function, because it does not establish the basic condition for recovery of the muscles. Strabismus surgery should not be performed as well during the decompression of the orbit because of a real possibility of spontaneous recovery of the function of the extraocular muscles after the decompression. If spontaneous recovery, supported by exercises of eye movements, does not occur three months after the operation, a strabismus operation is indicated. The first day after orbital decompression, it is necessary to start with exercises of eye movements to prevent fibrosis, scar around extraocular muscles which can limit the function of the muscles.

The described method of orbital decompression is logical, based on an understanding of the pathology, is less hazardous, is relatively easy to perform, gives constantly good functional and aesthetic results and allows rapid recovery.

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